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SCHIZOPHRENIA ACROSS CULTURES:
SIGNIFICANT INTERACTIONS OF BIOLOGY AND ENVIRONMENT

by

Kimberly Wilbanks

B.A., University of Missouri-St. Louis, 2007

A Research Paper

Submitted in Partial Fulfillment of the Requirements for the
Master's of Arts

Department of Anthropology
Southern Illinois University Carbondale
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SCHIZOPHRENIA ACROSS CULTURES:
SIGNIFICANT INTERACTIONS OF BIOLOGY AND ENVIRONMENT

By

Kimberly Wilbanks

A Research Paper Submitted in Partial
Fulfillment of the Requirements
For the Degree of
Master's of Arts
in the field of Anthropology

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TITLE: SCHIZOPHRENIA ACROSS CULTURES: SIGNIFICANT INTERACTIONS OF BIOLOGY AND ENVIRONMENT

MAJOR PROFESSOR: Dr. Robert Corrucini

Is schizophrenia caused and ultimately guided by biology or do environmental factors such as culture and interaction style impact the course and even the onset of this disease?

In this paper, the biological mechanisms of schizophrenia and the environmental influences on its behavioral and biological symptomology will be explored. Evidence will be provided that supports both the biological etiology of this mental illness as well as the strong influence of environmental factors, such as culture, on the severity, onset, and relapse rate of schizophrenia. It will be shown that even the structure and function of the schizophrenic brain is influenced by environmental factors, particularly social factors. The biocultural study of schizophrenia cross culturally is a fertile area and ideas for future research will be explored.

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INTRODUCTION

Schizophrenia is a psychiatric disorder with a strong biological basis that is also strongly impacted on several levels by cultural influences. The heritability of schizophrenia is well established in twin studies, fetal examinations of the developing brain, and genetic mapping (with at least 10 autosomes identified). Even though this disorder is biologically based, this does not mean that environmental influences from prenatal hormone exposure to beliefs systems and coping skills should be ignored. The human brain is a highly plastic organ that changes constantly in response to the activity of the person and the environment in which he/she lives (Weinberger & McClure, 2002). In this paper I will examine the areas of intersection between biology and culture to argue against a purely biological framework and to demonstrate to the reader the ways in which this mental illness and the lives of the individuals with it are impacted by social environmental factors.

Schizophrenia is characterized by symptoms such as hallucinations, delusions, disorganized thoughts, flat affect (lack of facial expression), paranoia, blunted emotions, asocial behavior, and many cognitive deficits. It is a chronic psychotic disorder that can be continuous from onset, which is usually between adolescence into early adulthood, until old age or death. However, it can be expressed as acute episodes punctuated by asymptomatic periods. Schizophrenia can be very heterogenous in expression, but the most common characteristics of the disorder are an inability to distinguish external reality

from internal thoughts and hallucinations, delusions, blunted affect, and social withdrawal. Most hallucinations are auditory, uncontrollable, and are abusive to the person experiencing them. Schizophrenic individuals in all cultures have difficulty relating to other people in socially appropriate ways and are prone to delusional beliefs that run contrary to the cultural values and beliefs of the people around them (Noll, 1983).

It would be a mistake to simply assume that these hallucinations, delusions, and the schizophrenic person's interpretations of her experiences are cut off from the effects of her upbringing and the culture she lives in. A schizophrenic person who was raised Christian is not likely to believe that Krishna or the Buddha speaks to him, because he was not raised with these symbols. Among the Iban in Borneo, there is no cultural conception of thought insertion or manipulation, even in interactions between spirits and human beings (Barrett, 2004). Clinical questions of thought insertion, even when translated in a culturally sensitive way, are confusing and nonsensical to many Iban individuals. Thus, the common schizophrenic symptoms of thought insertion and thought disorder are surprisingly lacking among the Iban (Barrett, 2004). However, is this because psychosis among the Iban is a qualitatively different disorder or because of different cultural conceptions of 'thinking' and 'feeling' and the different ways these cultural conceptions shape the schizophrenic individuals experiences? Focusing too narrowly on the biological nature of the disease ignores the impact culture has on the perception of the disease and the schizophrenic individual's experiences. It is, in fact, impossible to be completely outside of culture, and it is important to recognize that the

definitions of schizophrenia and the individual experiences of schizophrenics are culture-bound phenomena (Lucas, 2004).

The main questions of this paper are: Can the influence of the social environment and the larger culture have positive and negative effects on the course and severity of the illness and the quality of life of people with schizophrenia? And how does the experience of the external environment interact with the neurobiology of the schizophrenic brain? Throughout I will show the reader many ways in which culture strongly influences the symptomatic expression of the disease, neurobiological changes, and the experiences of schizophrenic individuals. To begin, I will present the first large scale evidence for cultural influence from the World Health Organization's (WHO) studies which examined the symptoms of thousands of individuals across several countries spanning over 30 years. These studies found striking and resilient evidence that schizophrenics in 'developing' countries actually had milder symptoms and fewer relapses compared to their peers in 'developed' countries. These findings left researchers searching for the underlying environmental and social reasons behind them.

In order to understand how the environment can influence the brain, I will then discuss the biological studies on the plasticity of the brain and the morphological and functional differences between schizophrenic and non-schizophrenic brains. I will also highlight the ways in which genetics alone are not enough to cause the onset of schizophrenia, but that the interplay between biological factors, such as hippocampal and stress cascade malfunction, and environmental stressors and coping mechanisms are what may ultimately determine whether the disease manifests within an individual or not. This same malfunctioning system also has a strong influence on the subjective experience of

schizophrenics and must be taken into account when considering effective treatment regimes and styles of interaction. It is necessary to explore how the brain reacts to changes in the physical and social environment around an individual and how this can shed light on how the cultural environment affects the symptomology of schizophrenia.

The remainder of this essay will concern the examinations into interaction styles within families and larger cultural effects conducted by psychiatrists, psychologists, and anthropologists. These discussions will demonstrate the many ways that the family environment and the larger cultural context can strongly influence the course and severity of schizophrenia. Throughout this section, I will tie together the ways in which behavioral symptomology and subjective experience can also be related to biological changes in the schizophrenic brain.

WESTERN BIOMEDICAL STUDIES DISCOVER CULTURE

The Western biomedical model tends to see disease in a purely biological manner and mental illness is no exception. The most common treatment for schizophrenia in the United States and Western Europe is drug therapy, which is used to control the biological cause of hallucinations and delusional behavior. Schizophrenia is viewed as a biologically based disorder with a biological course that is relatively permanent and stable throughout the life of the person. Therefore, from this viewpoint stabilizing the behavior of schizophrenic people through drug treatment is the best approach to improve their quality of life and help them to function in society. There is also a strong emphasis on control of these individuals that is historically grounded, which often subsumes the focus on treatment and improving the life of the mentally ill people in favor of conformity of behavior (Cutcliffe & Happel, 2009; Foucault, 1965).

Attempts to control the disruptive behavior of the mentally ill are not recent inventions. Within Western culture there is a long history of power and coercion concerning the mentally ill in which laws were created in order to 'put away' those deemed to be crazy into asylums and mental institutions. The names of these institutions have changed over the years, but the emphasis on controlling the behavior by removing the person from society has not. In fact, the discipline of psychiatry grew out of this historical framework of power and control, both as institutions to control the actions of

people in general and medical rules of diagnosis and therapy which guided discourse (Cutcliffe & Happel, 2009; Foucault, 1965).

More and more the rights of the mentally ill have been recognized, but it is also false to see the institution of mental health as merely a tool for the medical treatment of patients. Mental healthcare, particularly in the United States and other Western cultures, is most concerned with “controlling the uncontrolled” (Wilce, 2004: 359). Uncertainty about health creates a great deal of anxiety within people. Mental healthcare is an arrangement created by a cultural system in order to control the perceived chaos inherent in illness, particularly mental illness. Through legislation, psychiatrists are given the power “to detain patients and to force them to take powerful drugs” (Bracken & Thomas, 2001:725) as well as place them into seclusion and restraint for behaviors that the psychiatrists deem dangerous. These larger cultural factors within the United States, and many Western countries, create a world for schizophrenic individuals which is far different, and I would argue more hostile and oppressive, than the world that most non-mentally ill citizens experience. Undeniably, our medical model of mental illness which focuses almost solely on biological causes and treatment has been used to ignore a very real difference in the social and economic experience of schizophrenic individuals. This myopic focus of mental illness could cause (and probably has caused) many researchers in the field of mental health to ascribe purely biological causes to behaviors, and even morphological and functional changes in the brain, that are strongly influenced by the environment.

However, regardless of the reasons behind our mental healthcare model, this narrow and unquestioned emphasis on biology was shaken when the World Health Organization

(WHO) conducted the first large scale cross-cultural studies of schizophrenia ever conducted. The first two studies were the International Pilot Study of Schizophrenia (IPSS, 1967) and the Determinants of Outcome of Severe Mental Disorders (DOSMeD, 1978). Both were funded and coordinated by the WHO starting in 1967 and together they spanned 19 countries, 30 research sites, and thousands of people diagnosed with schizophrenia (Jablensky et al., 1992; Hopper & Wanderling, 2000). It was found that schizophrenic patients living in “developing” countries had a much better prognosis and higher rates of remission than patients living in so-called “developed” countries like the United States and those in Western Europe (Jablensky et al., 1992; Hopper & Wanderling, 2000; Watters, 2010). What’s more, the percentage of individuals with schizophrenia who were judged to be severely impaired were higher in developed countries (40% of the schizophrenics) than in developing countries (24%) (Watters, 2010). This is very ironic considering the fact that assumedly schizophrenic people in “developed” countries have access to better treatment and more stable living conditions.

This study sparked a debate that continues to this day. Later studies using similar methods have expanded on the research pool and longitudinally assessed some of the original participants. Interestingly, their findings have largely supported those of the original WHO studies. The International Study of Schizophrenia (ISoS, 1997), another WHO collaborative project, was a particularly salient study conducted by Kim Hopper and Joseph Wanderling (2000) beginning in 1997. The focus of the study was simply to examine the validity and robustness of the original WHO studies’ findings after 13 years and, if the original findings were supported, then they examined if there were any confounding variables that could have accounted for the difference which are unrelated to

the effects of culture. They examined 809 people in 13 research centers and 11 countries, many of whom were the same people from the original WHO studies. In addition, they also examined a separate sample from two different regions; Hong Kong and Madras. Their findings strongly supported the original findings of the WHO study and they determined that the potential sources of bias were not strong enough to justify the great difference in the course and prognosis of the disease in developing countries.

Even though these studies were groundbreaking and strongly pointed to large cultural and regional influences, the debate still rages as to what exactly caused these results. Many people still argue about the validity of the original WHO results from a variety of different points of view. Even Hopper in a 2004 article noted more limits to the studies. The original WHO studies did not carefully define the term ‘culture’, instead the architects of these studies simply assumed that region or nation were largely synonymous to culture (Hopper, 2004). Thus, they lumped all of India under the heading of one culture, which is a serious issue because India represents a multitude of cultures and regions with varying levels of development and economic stability (Hopper, 2004). The complexity of cultures and local variations even within cultures can be enormous and difficult to control for when trying to limit variables and discern what has the most impact on mental disorders. However, Hopper points to several intriguing themes of interconnectivity, such as expressed emotion and community incorporation, which can be seen to have a positive impact cross-culturally and have been seen to strongly mirror the WHO findings (Hopper, 2004).

Some researchers, particularly in the psychiatric community, argue that the social inequality, poverty, and violence within many developing countries in Africa, Asia, and

Latin America are not conducive to positive outcomes for individuals suffering from schizophrenia. Patel and colleagues (2006) argue that there is little evidence that socio-cultural factors actually improve the prognoses of schizophrenic people in developing countries and they question the results of the WHO study. They claim that romanticizing “under-developed” areas is harmful to individuals with this disease because it adds to their suffering and obscures the real hardships they face. Therefore, they contend that the results of the WHO studies were artifactual. They argue that the mortality rates for schizophrenics in developing countries are very high with many of them attributed to suicide. Also, the inpatient psychiatric care in many developing countries can be abysmal and resemble torture more than healthcare. They discuss conditions of severe abuse and neglect which have been documented in India and many countries in East and South Asia. Overall, they argue that there is evidence that points to a very negative view of mental illness in developing countries (Patel, 2006). They paint a very negative picture of developing countries and while it is true that many human rights violations occur in many of these countries, they do not address why the original WHO findings remained stable from the late 60s to the early 90s.

INTO THE GRAY MATTER

In order to fully understand how culture impacts the course of schizophrenia it is necessary to begin at the level of biology to discern how schizophrenic (and preschizophrenic) brains differ morphologically and functionally and how the environment impacts them differently, and similarly, to the brains of healthy individuals. In this section, I will explore studies which focused specifically on the plasticity of the brain and how the environment schizophrenic individuals live in and their perceptions of it can have an almost direct impact on several characteristics of brain chemistry and even morphology (Fitzgerald et al., 2004; Ho et al., 2008; Weinberger & McClure, 2002). I will then discuss articles addressing how differences in the preschizophrenic brain, such as abnormal hippocampal volume and function and a dysfunctional hypothalamic pituitary adrenal (HPA) axis, interact with the environment in which a preschizophrenic individual develops that can lead to the onset of this psychotic disorder (Walker & Diforio, 1997; Corcoran et al., 2003; Thompson, Pogue-Geile, & Grace, 2004; Walker et al., 2010). An analysis of a malfunctioning stress cascade and hippocampus interacting with the environment contributes to our understanding of how cultural factors can have such a large impact not only on behavioral symptomology, but also on brain morphology.

The Brain Meets the World

Many studies have found consistent differences in brain plasticity and cortical inhibition between schizophrenic subjects and normal subjects (Fitzgerald et al., 2004; Ho et al., 2008; Weinburger & McClure, 2002). Plasticity (or neuroplasticity) occurs when the brain changes structurally in order to adapt to new stimuli. Growing or pruning dendritic and axonal connections, increasing myelination in the connections that need faster transmission, and increasing or decreasing the number of receptor sites on a neuron are all ways that the brain responds plastically to new demands. Cortical inhibition is when the brain stops or slows neuronal firing after stimulation. Reduced cortical inhibition means that the brain has less of an ability to stop neurons from firing. This can cause a cascade of ‘run away’ firing which is part of the mechanism in the frontal and temporal lobes that turns into what we identify behaviorally as hallucinations and other thought disturbances. This reduced plasticity and abnormal inhibition could impact the “capacity of brain systems in these patients to respond to new stimuli” (Fitzgerald et al., 2004: 23).

Plastic responses over time in schizophrenics are “changes in synaptic activity, increases in dendritic length, changes in spine density, synapse formation, increased glial activity, and neurogenesis” (Fitzgerald et al., 2004: 18) which have been shown to differ significantly from non-disordered subjects. Postmortem studies of schizophrenics have found irregularities in “adaptive cellular processes” (healthy maintenance of cell function) within neural components. Postmortem studies have also shown abnormal sprouting patterns of axons, which transmit the action potential (i.e. information) from the cell body to the dendrites of other cells, and the disordered growth of the synapses, which are the points of communication between neurons. Neural functions that control

long-term potentiation (LTP) and long-term depression (LTD), which are involved in learning and memory, are abnormal in postmortem studies of schizophrenics (Fitzgerald et al., 2004). These are only a few of the differences found in schizophrenic brains. Most of this information is gotten from postmortem studies, so it is unclear whether all of these changes are a result of the disease process or a combination of the disease process and environmental factors.

So how do we distinguish those degenerative processes impacted by the environment and those specifically caused by the disease process? Many researchers have argued that the decreases in brain plasticity seen in schizophrenic individuals are simply an indication of the disease's impact on the brain and not environmental factors.

Fitzgerald and colleagues conducted a study to determine the nature of the abnormalities and any differences that exist between medicated and unmedicated schizophrenic individuals. In order to explore brain plasticity in living subjects, Fitzgerald and colleagues conducted a study using transcranial magnetic stimulation (TMS) techniques to measure cortical inhibitory activity and brain plasticity among 26 schizophrenic subjects and 18 control subjects. They measured their results using MRI scans. The results indicated several key differences in brain plasticity and cortical inhibition between schizophrenic subjects and controls. TMS lengthens the period of the cortical silent period (CSP) which is indicated by the suppression of tonic motor activity. A greater reactivity to TMS indicates plasticity, which means that the more plastic the response the longer the silent period between firings would be. As expected, the CSP was longer for the controls than the schizophrenic subjects (Fitzgerald et al., 2004).

Overall, there were consistent significant differences in measures of brain plasticity and inhibition. Reduced inhibition scores among the schizophrenic groups was highly correlated with reduced plastic response. The authors suggest that this reduced plasticity and abnormal inhibition could impact the “capacity of brain systems in these patients to respond to new stimuli” (Fitzgerald et al., 2004: 23). Reduced plasticity could also have implications for explaining decreasing brain volume and the enlarging ventricles of schizophrenic individuals over time, because reduced plasticity could impact the preservation of cellular integrity. Also, reduced plasticity could explain the deficits in working memory found in schizophrenics and, interestingly, these abnormal plastic processes (which are largely of genetic origin starting in childhood) could also have implications for the development of psychoses (Fitzgerald et al., 2004: 23).

Ho and colleagues (2008) examined the functional and structural abnormalities in schizophrenic brains. The authors conducted a 10 year longitudinal study of 73 schizophrenic patients and 23 controls using MRI scans to examine the structure of the brains. They sought to establish whether the enlarged ventricles and brain volume changes in schizophrenia were a normal result of aging or were indicative of the disease process. Ventricles are the spaces in the brain that this study, and many MRI studies, found enlarge over time in schizophrenic patients (Ho et al., 2008; Weinburger & McClure, 2002). Ho and colleagues also sought to ascertain the effect that changes in brain volume could have on the behavioral and mental functioning of schizophrenic individuals. They found, early in the course of schizophrenia, there was a progressive increase in the size of the cortical sulcal cerebral spinal fluid (CSF) spaces. Unlike controls, the schizophrenic subjects exhibited a progressive decrease in the volume of

white matter in the frontal lobe which was connected to an increase in CSF volume in the frontal lobe. White matter is made up of the connective fibers that facilitate communication between cells, the axons and dendrites. White matter does not consist of the neuronal bodies themselves, which is termed grey matter. Brain volume changes in white matter were most evident in the frontal lobe and are connected to functional impairment, such as a more severe presentation of negative symptoms (i.e. flattened affect, slowed cognitive functioning, and asocial behavior). Reductions in white matter in the frontal lobe were associated with diminished executive functioning and those with greater lateral ventricular enlargement over time had poorer outcomes. Control subjects exhibited an increase in frontal lobe white matter volume over time, which is the converse of what happened to the schizophrenic patients. According to Ho and colleagues (2008:592), this indicates “a dysmaturation process in schizophrenia, in which white matter fails to undergo the usual trajectory of progressive myelination or increased interconnection”.

I find it interesting that the schizophrenic subjects with the most chronic and severe forms of this illness “were more likely to show longitudinal alterations in brain volumes” (Ho et al., 2008: 592). It would be interesting to see if changes over time like this inevitably occurred or if this process could indeed to be slowed down by environmental factors like culture and/or treatment method. The study showed that medications had no effect on the “progressive volumetric brain changes” (Ho et al., 2008:593) in spite of the fact that they behaviorally controlled many symptoms. What I find most interesting is their discussion of the fact that the brain responds to environmental effects and that some of their findings were not merely connected to

neurodevelopmental features, but also to the influence of “chronic illness, including poor nutrition, fluid imbalance, and diminished social and environmental stimuli” (Ho et al., 2008:593). They only discussed these environmental influences cursorily, but this very statement suggests that a more comprehensive examination of white matter fluctuations and environmental influences is necessary.

These two studies, and others like them, do not really discuss the long term effects of medication use and the emotional and social effects of schizophrenia and its treatment. Also, most of them are almost exclusively conducted by and on people in the United States and Western Europe. Thus there is a similar philosophy underlying them. They only cursorily examine the social situations of many of their subjects and they do not address the large difference between the social and environmental experiences of their schizophrenic subjects and the normal controls.

So do environmental factors have an impact on plasticity and cortical inhibition? Fitzgerald and colleagues (2004) have already shown that decreased plasticity and reduced cortical inhibition lead to abnormal brain morphology and function, but the question is do factors such as medication, different treatment methods, and familial and other social interactions have an effect on plasticity? Postmortem studies can only show us the end result of the interaction between environmental and biological factors, but not what factor (or combination of factors) contributed most to the morphological and functional differences in the schizophrenic brain. MRI studies, even with TMS, only provide a snapshot into what is happening in the brain at any given period of time. Unless long term longitudinal or cross sectional MRI studies are done, they cannot tell us about changes over time and what contributes to them.

Perhaps the best report of plasticity and the schizophrenic brain was the metastudy conducted by Weinburger & McClure (2002). They performed an extensive review of the use of MRIs to measure changes in the schizophrenic brain and found a huge degree of variation between studies which had not previously been addressed. They point out that the results of longitudinal studies of cognitive function in schizophrenic individuals do not support progression of degenerative processes for at least the first 20 years of the illness. They point out that MRI measurements can vary widely due to “differences in image acquisition and analysis techniques, alterations in neuronal and nonneuronal tissue compartments, physiological alterations in brain tissue (e.g. changes in tissue perfusion, fat, and water content), and changes in other chemical constituents that make up living brain” (Weinburger & McClure, 2002:556). Also, there is the simple fact discovered in studies of healthy and unhealthy brains that the shape of the brain is greatly affected by the environment and the quality of life of the individuals. Rapid changes in volume measurements can result from decreases or increases in body weight, alcohol or drug use, and hormonal concentrations. Also, ventricular size can fluctuate greatly within just a few months. Because of these rapid fluctuations, changes in ventricle size seen in the MRIs of schizophrenic individuals might not reflect degenerative processes but rather normal fluctuations caused by environmental changes. The facts of life for many schizophrenic individuals, such as joblessness and homelessness and lack of social interaction, could contribute largely to the fluctuations in white matter and cortical size seen in MRIs. These environmental factors have largely been ignored by the psychiatric community because the focus is narrowly on the disease process and the genetic and biological processes related to it (Weinburger & McClure, 2002). Focusing on these processes of

cortical and ventricular shrinkage and neuroplasticity with respect to different cultural contexts and treatment regimes might shed light on how much of this degeneration is a product of the disease and how much of it is a product of treatment regimes and social stigmatization, for instance in Western biomedical institutions and Western culture in general.

The (Dys)functional Brain Under Stress

Another exciting area of research into the schizophrenic brain examines the impact that prenatal and early childhood hippocampal dysfunction has on the onset of schizophrenia, particularly in relation to the hypothalamic pituitary adrenal (HPA) axis. Schizophrenic individuals have smaller hippocampal volumes, smaller prefrontal cortexes with fewer synaptic connections, and have significantly higher basal cortisol levels than nonschizophrenic controls (Walker & Diforio, 1997; Corcoran et al., 2003; Thompson, Pogue-Geile, & Grace, 2004). These researchers argue that the altered neuronal and dendritic structures in the brains of preschizophrenics can be further altered by a malfunctioning stress response system and hippocampus. This can ultimately lead to damage and a dysfunctional reorganization that culminates in onset of psychotic symptoms and a long-term psychotic mental disorder.

Prodromal behavioral symptoms have been recognized in preschizophrenic individuals. These prodromal symptoms indicate the future onset of psychotic illness which is reliable 40 to 50 percent of the time (Corcoran et al., 2003). Prodromal symptoms often “occur months to years before the full onset of the syndrome” typically beginning in early to middle adolescence and “include depression, increased anxiety, difficulties in concentrating, changes in cognition and perceptions, distrust, social

withdrawal, anhedonia, and deterioration of function” (Thompson, Pogue-Geile, & Grace, 2004: 875).

It appears that there is an association between the behavioral indicators in the prodromal period and certain biological markers. Walker and Diforio (1997) and Corcoran and colleagues (2003) argue that smaller hippocampal volumes and a malfunctioning HPA axis, indicated by heightened basal cortisol levels, are early biological markers for the onset of schizophrenia. Individuals with prodromal symptoms for schizophrenia have smaller hippocampal volumes than healthy controls. Corcoran and colleagues (2003) present evidence that “abnormality in the hippocampus may be one feature of the neural diathesis in prodromal patients that puts them at risk for developing psychosis in the context of stress” (674). The hippocampus of a prodromal individual functions abnormally and at a reduced capacity and has a diminished size. This abnormality can stem from genetic factors, prenatal and early childhood disturbance, or a combination of both that results in “an unrestrained HPA axis response to stress, as the hippocampus is rich in GC (glucocorticoid) receptors that are integral to the negative feedback of the HPA axis” (674).

The HPA axis in healthy individuals prepares the body for stressful situations by increasing heart rate and focusing attention. When the stressor is no longer present, a negative feedback system initiated by the hippocampus works to decrease the level of stress hormones in the body and helps the individual to relax. When an individual is presented with a stressor, the hypothalamus in the brain releases corticotrophin releasing hormone (CRH) into the bloodstream which is picked up by the pituitary gland, which then is stimulated to release adrenocorticotrophic hormone (ACTH) into the blood. ACTH

travels to the adrenal glands that cap the kidneys and induce them to release glucocorticoids (GC), otherwise known as stress hormones. The primary stress hormone in primates is cortisol (Thompson, Pogue-Geile, & Grace, 2004).

In a healthy system, when cortisol reaches a certain saturation in the blood, GC receptors on the hippocampus would bind with cortisol and initiate a negative feedback system which induces the hypothalamus to stop secreting CRH, which would stop the release of cortisol from the adrenal glands. All the remaining cortisol would eventually be broken down by enzymes in the bloodstream. However, the smaller hippocampuses of prodromal and schizophrenic individuals have fewer GC receptors and do not adequately initiate this negative feedback system. Therefore, their bodies maintain a higher concentration of cortisol in the blood because the hypothalamus is never stimulated to stop releasing CRH. This leads to a more extreme reaction to stressors and less of an ability to cope with new stressors (Corcoran et al., 2003).

However, a diminished capacity to deal with stress is only the tip of the iceberg. Chronically high levels of glucocorticoids are toxic to the hippocampus and lead to an even greater reduction of GC receptors on the hippocampus and in other brain regions. They also produce a number of neural changes that result in a “sensitization to stress” and create dysfunction in many different systems (Corcoran et al., 2003: 673), particularly in the dopaminergic systems of the prefrontal cortex and the subcortical region of the limbic system that are implicated in psychotic perceptions and behaviors (Walker & Diforio, 1997; Thompson, Pogue-Geile, & Grace, 2004). Chronic stress has been shown to lead to cell death in the hippocampus, which creates permanent changes, as well as “potentially reversible processes, such as atrophy of dendrites on excitatory pyramidal neurons,

decrease in the generation of new neurons, reduced expression of neurotrophic factors such as brain derived neurotrophic factor, and suppression of long-term potentiation, the biological underpinning of memory” (Corcoran et al., 2003: 673). Here we see connections between reduced plasticity and white matter shrinkage with a malfunctioning hippocampus and a run-away stress response system. Stress, even in non-schizophrenic individuals, can not only sharply prune back nerve fibers, but can also inhibit the cyto genesis of neurons and the healthy activity of many brain regions. In other words, it damages the brain and decreases its ability to adapt to new stimuli, hence reduced plasticity.

Dysfunction in the hippocampus is not the only malfunctioning system present in the preschizophrenic brain which is exacerbated by cortisol. Dysfunction also exists in the dopaminergic systems, particularly the prefrontal cortex and subcortical regions in the limbic system, which serves to inhibit healthy HPA axis feedback and intensifies dysfunctional change in these regions (Corcoran et al., 2003; Thompson, Pogue-Geile, & Grace, 2004). The dopaminergic systems, alongside the hippocampus, fail to begin the negative feedback system in the HPA axis resulting in decreases in GC receptors and many other structural changes.

So what does this have to do with the interaction of biology and culture? Walker and Diforio (1997) were the first to argue that it is this biological malfunctioning which underlies the onset of psychosis. They argued that if the stress response could be controlled at the critical prodromal period in adolescence, that schizophrenia onset may be completely avoided in some individuals. Exposure to stress and symptom exacerbation in schizophrenic individuals has already been demonstrated in many neurobiological

studies (Thompson, Pogue-Geile, & Grace, 2004; Walker et al., 2010) as well as psychosocial naturalistic studies (Levene et al., 2009; Lopez et al., 2009; Rosenfarb, Bellack, & Aziz, 2006; Jenkins, 1991). However, the question is whether or not the dysfunctional systems themselves inevitably increase cortisol levels in schizophrenic individuals that result in psychotic onset (regardless of environmental stressors) or if environmental stressors, and perhaps unproductive coping behaviors, interact with the dysfunctioning system that then results in the onset of schizophrenia. Both Corcoran and colleagues (2003) and Walker and Diforio (1997) argue that it is a combination of the dysfunctioning systems, developmental increases in cortisol and overall changes in the structure of the brain during adolescence, behavioral coping mechanisms, and external stressors that determine whether the person will develop a psychotic illness or not. Because of a dysfunctional HPA axis, hippocampus, and dopaminergic system, prodromal individuals are more vulnerable to stress (Corcoran et al., 2003). Thus they respond more intensely to everyday stressors and have difficulty calming down. Coping mechanisms that help to alleviate stress, finding ways to decrease overall stressors in the environment, and family members who work to adapt their behaviors to account for the enhanced stress response of a prodromal, and usually adolescent, individual may make a huge difference in the development of their brain and, ultimately, whether or not they become schizophrenic. Familial interactions and coping behaviors also make a huge difference in the severity of symptoms and quality of life for individuals who are currently schizophrenic, which I will discuss in the next section.

THE IMPORTANCE OF INTERACTION

Many researchers (Levene et al., 2009; Lopez et al., 2009; Rosenfarb, Bellack, & Aziz, 2006; Jenkins, 1991; McGruder, 2004; Sullivan, Allen, & Nero, 2007) decided to examine how the course of schizophrenia varies across cultures and what effect the environment and the cultural context has on the symptomology and quality of life of schizophrenic individuals. The ultimate hope for all researchers is to eventually approach the best model for the treatment of schizophrenia and to improve the quality of life of schizophrenic individuals. Many studies have been conducted cross-culturally that examine different interactional styles and how they relate to culture, the illness severity, and rates of relapse in schizophrenic individuals. No single interactional factor has been found to be more salient than that of the level of expressed emotion between family members and their schizophrenic relatives (Levene et al., 2009; Lopez et al., 2009; Rosenfarb, Bellack, & Aziz, 2006). These studies are most often conducted by psychologists and psychiatrists, but more and more anthropologists are becoming interested in expressed emotion because of its high degree of cross-cultural consistency (Jenkins, 1991). Finally, many anthropological ethnographies (Jenkins, 1991; McGruder, 2004; Sullivan, Allen, & Nero, 2007) have been conducted that focus particularly on the lives of schizophrenics and their families in specific cultures. They bring together many aspects of the psychological and biological studies, but also try to understand cultural

practices and belief systems that contribute to cultural differences in understandings of and treatments for mental illness.

Interactions of Significance

In answer to the WHO studies findings, many psychologists and psychiatrists have increasingly zeroed in on interactions between schizophrenics and their primary caretakers, which are usually members of their families. What they found most consistently was that specific interaction styles characterized by attitudes of hostility, criticism, hypervigilance, and emotional overinvolvement had the most reliably negative effect on schizophrenic individuals. This interaction style was termed high expressed emotion (EE), because it is characterized by an increased amount of emotional involvement toward the schizophrenic family member, expressions of anger and hostility, and an increased number of critical verbalizations. Expressions of negative attitudes and verbalizations tend to vary, but in general they have been found to be related to expressions of delusional thinking or other behaviors that the family members consider odd and inappropriate. Conversely, low EE family members are characterized by a more emotionally even reaction to their schizophrenic relatives. They tend not to focus on or try to control the strange verbalizations and actions of their relatives and they also tend to give their relatives space when they become upset. Patients who live with high EE family members are two to seven times as likely to relapse as those who live with low EE family members (Levene et al., 2009; Lopez et al., 2009; Rosenfarb, Bellack, & Aziz, 2006; Walker & Diforio, 1997; Watters, 2010).

However, the vast majority of studies involving expressed emotion have been conducted between ethnic groups in Western societies. In general, it has been found that

white American families exhibited significantly higher EE than Mexican Americans and African Americans toward their schizophrenic family members, regardless of socioeconomic status (Lopez et al., 2009; Rosenfarb, Bellack, & Aziz, 2006) and this has been associated with greater expression of symptoms and a higher relapse rate in their schizophrenic relatives.

So what exactly causes this difference in interaction styles and why would family members react in these ways towards their relatives? In reality, most high EE family members believe they are helping their relatives. There are some cultural reasons for the difference between ethnic groups and some reasons behind the differences within ethnic groups. Rosenfarb, Bellack, and Aziz (2006) sought to understand the cultural dimensions that cause this difference, particularly on how caregivers perceive their burden of care, react to psychosocial stressors, and the way this relates to how they view the behavior of their schizophrenic family members and their roles as caretakers. They discovered that cultural factors definitely impact the extent to which schizophrenic family members were viewed as burdensome as well as their relatives' negative attitudes towards them. African American families were much more likely than white families to see their role as caretaker as an important and valuable one. They were also much more likely to utilize community support (usually that of a church and extended family) in caring for their schizophrenic relatives and to conceptualize their role in spiritual terms. African American family members were also more likely to view their relatives' disease in spiritual terms rather than view the illness as solely biological in origin and the spiritual view often overlapped with a biological understanding. Conversely, white families were less likely to use community support (or have as much access to it) and

they were more likely to describe their relatives' illness in biomedical terms. Within interactions, white family members responded more negatively to the odd behaviors and thoughts of their relatives during discussions, which was associated with high EE attitudes. The number of odd behaviors and verbalizations by their schizophrenic relatives were associated with an increased perception of burden and an increase in intrusive behaviors, such as critical and 'corrective' remarks and close monitoring of their relatives' behavior. High EE among white family members was connected to attempts at controlling the behavior of the schizophrenic individuals and beliefs that people "can control their own lives" (158).

Levene and colleagues (2009) also found that the more negatively a relative rated the behavior of the schizophrenic individual, the worse their outcomes would be. They go on to elaborate that "specific reactivity and perceived burden of relatives in response to symptoms and illness behavior of the patient may synergistically interact to potentiate psychotic symptoms for the patient" (449). In other words, the more a schizophrenic relative acted out "illness behavior", that was viewed and reacted to negatively by family members, the more their family members felt burdened and reacted in more hostile and critical ways. This could create an interaction feedback loop where the reactions of the family member would increase the stress of the schizophrenic relative and increase the severity of their symptoms, which would in turn increase hostile and controlling behaviors of the family members until the point that the schizophrenic individual relapses and is readmitted to the hospital.

This can be viewed in not only cultural and behavioral terms, but also biologically. The hippocampal and HPA axis malfunctioning in the schizophrenic brain which initiates

onset of the disorder and causes structural and functional changes in many brain systems is exacerbated by a run-away stress response. Because of the cytoarchitectural changes in the schizophrenic brain, they experience stress more intensely and retain heightened cortisol levels for much longer than healthy individuals.

Corcoran and colleagues (2003) reported on several longitudinal studies that connect psychosocial stressors, variation in cortisol levels, and symptom severity in schizophrenia. They argue that there is a strong argument for a connection between symptom severity and relapse in schizophrenia with psychosocial stressors. In one study of schizophrenics admitted to a psychiatric ward due to acute psychotic episodes, researchers found a strong correlation between cortisol levels and the “severity of recent stressors” (679). Cortisol levels in schizophrenics are not only related to symptom severity, but are also strongly tied to “more stressful life events during the 3 weeks preceding a relapse than they do during other time periods” (679). Their findings indicated that psychotic relapse can be worsened or even caused by psychosocial stress.

Walker and Diforio (1997), Corcoran and colleagues (2003), and Thompson, Pogue-Geile, & Grace (2004) all discuss the ways in which psychosocial stress exacerbates symptom severity and relapse rates and how chronic stress can cause further damage to brain regions, including many cognitive deficits such as difficulty concentrating and memory loss. Stress not only exacerbates symptoms, but causes schizophrenics to increasingly isolate themselves as stressors become more and more difficult to cope with. Family interactions are often a primary contributing factor and when these interactions break down and contribute to relapse, there is a greater need for schizophrenic individuals to find alternative ways to decrease stress such as through withdrawing further and

reducing stimulus. As discussed earlier, schizophrenics also have reduced cortical inhibition and a diminished plastic response as well as greater ventricular volume and decreased white matter. Ho and colleagues (2008) discussed how decreased white matter in the frontal lobe is associated with greater negative symptom severity, which includes asociality. However, Weinberger and McClure (2002) pointed to ways that environmental factors contributed to decreases in white matter over time and how decreased plasticity could interact with a stressful and/or unstimulating environment to bring about white matter shrinkage. I argue that diminished plasticity, reduced cortical inhibition, a malfunctioning stress response, and a stressful environment interact to contribute to worse outcomes biologically, cognitively, and behaviorally in schizophrenia.

There is no argument concerning the damaging effects of stress on the schizophrenic brain and the individual's quality of life. They respond to stress more intensely and their brains are more greatly damaged by it than healthy individuals. Thus it is of great importance to insure individuals with schizophrenia live in environments in which stress is minimized and healthy coping skills are used and reinforced. The research into familial interactions is shedding light on how factors like expressed emotion and underlying philosophy can impact symptom severity and relapse rates in schizophrenia. In the next subsection, I will show how cultural factors guiding familial and community interactions can help decrease the severity of schizophrenic symptoms or exacerbate them.

Interactions of Culture and Meaning

So, what does expressed emotion mean cross-culturally? Is this a term that a psychologist equipped with the Western understanding of the behaviors and meanings

behind interactions could just pack off to another culture entirely and assess with families there? This is what often happens in cross-cultural studies of expressed emotions, which have been done in many different places other than the U.S and Western Europe, such as Italy, Mexico, China, Taiwan, India, North Africa, and Australia (Watters, 2010).

Janis Hunter Jenkins (1991) is one anthropologist who took social scientists (including anthropologists) to task for not really examining what expressed emotion is and whether it is being applied appropriately across different cultures. She argued that expressed emotion can be a powerful tool for assessing how culture and interaction styles could potentially shed light on what environments are best for schizophrenic individuals and can ultimately inform treatment. However, there are many problems with its use. The cultural context has to be taken into consideration when studying family interactions and schizophrenic illness.

Jenkins (1991) conducted an ethnography among Mexican-American families caring for schizophrenic relatives to examine interaction styles and illness severity and found that several factors had to be examined before any understanding of surface observations could be gained. For instance, what is considered intrusive behavior among Mexican-American families? Family members are said to be intrusive when they are so overly involved in their relatives' lives that they abandon other activities and interests (including outside employment and parental responsibilities toward other children) to focus on their ill relatives. These individuals also express greater sadness and anxiety toward their situations and the illness of their relatives than the norm. In the United States, a behavior that is typically labeled as intrusive is when a family member visits his/her relative every day and brings food to them when they have been admitted to the hospital. It is also

characterized by such behaviors as not wanting their relatives to leave the home or be unattended for any period of time.

In the more sociocentric environment of Mexican-American families, it is expected that families (particularly mothers) will visit any relatives daily in the hospital in order to make them feel more comfortable and to show their support. Also, a high amount of interaction with relatives in general is more the norm and it is not odd for a schizophrenic family member to be around at least one relative when they are at home or go out with them. Jenkins states that it is better to discern what is considered appropriate behavior by the culture under study than to use categories and notions informed by the researcher's cultural understandings that may have no meaning when applied to people whose cultural beliefs and practices are different. Jenkins learned to discriminate between behaviors which were considered acceptable and which were not from the reactions and thoughts of other family and community members. For instance, one woman who broke down crying and screaming every time she couldn't find her schizophrenic son and would never let him out of the house without her was viewed as having gone overboard by other family members (Jenkins, 1991: 408-409).

Another point of cross-cultural confusion is what constitutes criticism and what does not. Some statements might sound like hostile exchanges, but do not contain the anger or result in the same amount of stress as truly hostile interactions. There is a relative amount of expressions of anger that are tolerated more in some cultures than in others. Among British households, open expressions of anger and hostility are viewed as much more extreme than in Mexican-American households where some amount of venting is expected and therefore does not cause the same emotional reaction (Jenkins, 1991). These

cultural factors shape what family members view and, perhaps more importantly, what a schizophrenic individual would understand as interactions of criticism and hostility.

Finally, Jenkins' (1991) deeper study of the cultural aspects of interaction helped her to understand how different conceptions of mental illness and appropriate behavior related to the way families viewed and treated their relatives. This, in turn, impacted the severity of the schizophrenic family member's symptoms and stress level. She found that the concept of *nervios* was how most Mexican-American individuals conceived of schizophrenia, which influenced how they related to and thought of their schizophrenic relatives and, significantly, how the schizophrenic individuals viewed themselves. *Nervios* roughly translates to 'nerves', but it signifies more than the meaning most English speakers would attach to it. *Nervios* is a broad category that relates to various negative emotional and mental states. Schizophrenia is seen as a very extreme case of *nervios*. Mexican-Americans believe that "severe cases of *nervios* are not considered blameworthy, or within an individual's control, the person who suffers its effects is deserving of sympathy, support, and special treatment" (415). Mexican-American families responded to *nervios* by sympathetic inclusion rather than exclusion. There also is less of an emphasis on peoples' ability to individually control their lives and Jenkins related this to the socially-centered viewpoint in which all struggling family members should receive support.

Jenkins (1991) demonstrated the importance of examining interactions between family members cross-culturally and even the validity of examining expressed emotion, but she also pointed to many dangers of categorization and meaning creations that differed cross-culturally. The behaviors and interactions that are considered appropriate are intimately

shaped by the cultural context as well as how to respond to breaches in protocol. Also, the way madness in general is understood and the cultural and religious methods of adapting to it are highly significant to understanding interactions and how they influence the health of schizophrenic individuals.

Other anthropologists have examined interactions cross-culturally in a culturally conscious and relevant manner. Addressing the results from the WHO studies, Juli McGruder (2004) argues that in order to get at the meaning of their results, ethnographic studies need to be done among people in the developing world who live with family members with psychotic illnesses. This way a more detailed picture could be developed concerning how social factors could influence prognoses in psychotic illnesses. She studied three different families in Zanzibar, Tanzania to examine interactions between relatives and the underlying cultural and religious contexts that inform and shape them.

In Zanzibar, belief in spirit possession is almost universal and deflects a great deal of anxiety onto uncontrollable outside forces. Spirits are troublesome, desirous, and rude beings that possess people and cause them to behave in unacceptable ways. The most popular definition of madness is spirit possession and many rituals are done to appease and draw out spirits in afflicted people. Interestingly, traditional medicine and the Western biomedical treatments are both utilized by locals, frequently in equal amounts by the same families. An Islamic worldview, Western biotechnology, and traditional medicine coincide in belief systems and are not seen as contradictory (McGruder, 2004).

McGruder (2004) connects the belief in spirit possession to the view of the impermanence of madness and low expressed emotion. By seeing madness as transient and caused by outside factors, it means that schizophrenics in Zanzibar are not viewed as

being without hope for recovery, as they often are in the United States. When they are experiencing psychotic symptoms they are allowed to isolate themselves and not participate in household chores or meals. However, when they are doing well they are reincorporated into the social life of the family and are expected to participate in the daily work of the home. The illness is considered transitory and thus when the schizophrenic family member is doing better they are viewed as healthy or, perhaps more accurately, not possessed. McGruder asserts that, in general, emotional expressions of anger and hostility are strictly controlled by cultural and Islamic values and by the simple necessity of maintaining peace in large extended family households. Also the belief that accepting suffering in this life will lead to rewards in the afterlife is an important part of conceptualizing and coping with illness in this Muslim society.

McGruder (2004) demonstrates how these local and Muslim beliefs and practices are related to a relational style that is less critical and judgmental toward schizophrenic family members. She determined that the interaction style of families in Zanzibar is characterized by low expressed emotion because of the tendency to not attend to the strange behavior or even most outbursts by the schizophrenic relatives and to allow them to distance themselves if needed. Criticism happens, but it is usually in response to specific behaviors or needs (“I need you to find a job because we need money” or “look at this mess!”) rather than globalizing assessments (“lazy” or “worthless”).

Culture, Biology, and Prevalence

Both McGruder (2004) and Jenkins (1991) specifically examined familial interactions and how they are influenced by culture and how they, very particularly, affected the lives

of schizophrenic individuals. While small-scale ethnographies can flesh out and add depth to the understanding of interaction, they do not have the statistical power to show that cultural factors do have an impact on the expression of schizophrenia. Although, they do provide a great deal of support for the larger scale and shallower psychological studies that show an impact of interaction styles, specifically of expressed emotion, on the severity and rate of relapse among schizophrenics. Because of the intricately biological and cultural nature of schizophrenia, it is important to be able to connect these data to more large scale studies of the effect of culture on schizophrenia which is comparative and both culturally and biologically oriented.

Sullivan, Allen, and Nero (2007) sought to create a synthesis between bio-behavioral perspectives as well as examinations of the social and cultural factors that impact schizophrenic individuals in specific settings. As far as I can tell in my examination of the anthropological literature on schizophrenia, they are the first to conduct a biocultural study of this mental illness. It has been established that there is an underlying genetic cause to schizophrenia, but it is becoming increasingly clear that it is “also strongly influenced by epigenetic processes and environmental stressors” (189). There is an established worldwide 1% average prevalence of schizophrenia; however the authors state that the seeming uniformity of this figure obscures high degrees of variability between different regions. Recognizing this variability is important because those who back a narrowly genetic deterministic perspective for schizophrenia tend to emphasize uniformity. Ignoring variability means that many of the environmental factors that contribute to the disease’s expression are often overlooked.

Palau in Micronesia has an abnormally high prevalence rate of schizophrenia at 1.7%. What's more startling are the striking gender differences in prevalence. When separated by gender, women have a normal prevalence rate of 1%. Men, on the other hand, have a prevalence rate of 2.2%, which is over twice the average worldwide. Sullivan, Allen, and Nero (2007) studied the expression of schizophrenia in Palau in order to examine the reasons for this high prevalence among men. They used standardized clinical instruments to determine symptom levels and expression as a way to connect with the broader literature on the expression of schizophrenia and to provide a consistent basis for cross-cultural comparison. They also performed both a bio-behavioral and a cultural study in order to zone in on the most likely factors influencing the difference. They argued that studies within settings with extraordinary prevalence rates will help to shed light on "causal factors associated with schizophrenia in context" (Sullivan, Allen, & Nero, 2007:190).

I will not go into great detail about their bio-behavioral evidence here, but it really is not necessary because they found nothing biologically or psychiatrically distinct about schizophrenic individuals in Palau. The severity of schizophrenic symptoms was highly comparable to schizophrenics in the United States and there was also no significant difference in concentrations of positive or negative symptoms (Sullivan, Allen, & Nero, 2007).

Thus, they turned to the ethnographic data in order to try to understand the high prevalence of schizophrenia among men in Palau. Interestingly, schizophrenic women in Palau have a much higher rate of marriage than schizophrenic men and have the same number of children as the average Palauan woman. The marriage rate for men, on the

other hand, is only 10% and the average number of children per schizophrenic man in Palau was only 0.5 per man. This simple fact shows that there is a very real difference between the way mentally ill men are perceived and treated compared to women.

The researchers argue that this difference, as well as the unbalanced prevalence of the disease, is related to cultural differences in the way young men and women are treated. At puberty, Palauan boys have to leave their homes in order to observe strict incest taboos, whereas their sisters remain in the home for the rest of their lives with their husbands moving in with them and their families. Throughout adolescence, most boys have no stable home and instead move around between homes of different relatives. Young men have to establish their careers, home, and family before they are considered adults and treated with respect. Men move in with their wives' families at marriage, but still owe social and monetary obligations to their natal families as well as their wives' families. Meanwhile, the employment opportunities have decreased and many young men find it necessary to leave their country in pursuit of gainful employment. On top of all this, young men are still required to "observe and fulfill customary obligations" (Sullivan, Allen, & Nero, 2007: 200), even when their jobs take them far afield, which is a great source of stress.

Conversely, women have built-in responsibilities, security, and an established place in the household from childhood through old age. They have obligations to their families and are more restricted in their dress and movement, but they generally never have to worry about their security or place in the household. Their status is not dictated by their ability to find ephemeral and capricious employment, their work is domestic and generally proscribed by their life stage. They have identity and support within their natal

groups. Also, a greater amount of protection and warmth is shown to girls and women in Palau in general.

It is also no coincidence that the higher prevalence of schizophrenia among men in Palau is connected to an increase in social and political instability (Lowe, 2003; Sullivan, Allen, & Nero, 2007). In Palau, and most of Micronesia, it has become increasingly difficult for people (primarily men) to find work, which often forces them to search for employment in neighboring countries (Sullivan, Allen, & Nero, 2007). A disturbing new trend over the past thirty years in Micronesia is an increase in suicide rates and substance abuse, particularly alcoholism. What is especially striking is that “young men are at significantly higher risk for substance abuse, suicide, and diagnosed mental illnesses than are young women in Micronesia” (Lowe, 2003: 188). These correlations cannot be mere coincidence.

To some extent a young Palauan man has no meaningful identity until he earns an income, marries, has children, and engages in the matrix of social relationships and economic and political exchanges channeled through the brother-sister dyad and their respective spouses. In terms of social roles and responsibilities, those of young men are “floating” in comparison with the more proscribed roles of young women in Palauan society, which leaves them more vulnerable to a whole host of mental and emotional issues. This vulnerable phase in the lives of young Palauan men overlaps with the prodromal period between adolescence and late 20s in which the onset of schizophrenia most often occurs. During the same period of their lives women are not nearly as stressed as young men, which could go a long way to explaining the striking gender differences in the expression of schizophrenia.

Boys and men receive less instrumental, expressive, and global support than their female counterparts. Mentally ill women are much more likely to be cared for and protected by their families, but a mentally ill man might receive handouts of food from his family, but will not find shelter or much emotional support (Sullivan, Allen, & Nero, 2007). So much of a young man's status and identity is tied up in successfully navigating this period of his life and if he does not he will dishonor his family and will not be able to find a wife.

As I discussed earlier, high amounts of stress have been shown to influence the onset of schizophrenia as well as the expression of symptoms (Corcoran et al., 2003; Thompson, Pogue-Geile, & Grace, 2004; Walker & Diforio, 1997). Given the cultural evidence I have presented concerning the connection between familial interaction and larger cultural practices and beliefs with symptom severity in schizophrenia, it is not difficult to see that culture and biology connect in striking and, possibly, even measurable ways. The connection between chronic HPA axis activation, functional and structural changes throughout the brain, and the onset of schizophrenia can be understood at a more intricate and holistic level when one takes culture into account. Micronesian men have a much higher prevalence of schizophrenia than women in their culture because they encounter a greater amount of stress during the critical prodromal period. It might be too critical to say that this is a culturally sanctioned disparity, it is more likely that this is an inadvertent side effect of cultural practices and beliefs as well as larger economic and political influences that individuals have to live with everyday.

CONCLUSION

So can the influence of culture have positive and negative effects on the course and severity of the illness and the quality of life of people with schizophrenia? I have demonstrated many ways in which culture influences the expression of schizophrenia and could even influence whether or not the schizophrenic genotype (or the propensity for the disease) is expressed at all. There are many trends that have been identified that interact significantly with the severity, relapse rate, and even the prevalence of this mental disorder. The general picture that is being generated by psychological, psychiatric, and anthropological investigations on cross-cultural influences is that interactions within the family and within local communities can have a large influence on relapse rates and the severity of the disease. Hostile and critical interactions with emotional overinvolvement and hypervigilance have been shown to be highly correlated with the severity of schizophrenic symptoms and relapse rates. This has also been tied to heightened cortisol levels and worse neurobiological functioning. However, the problem is teasing out which came first, the interactions of high expressed emotion or the more severe symptoms of schizophrenia. More and more researchers are finding that there is a feedback loop interaction of worsening symptoms (alongside increasing basal levels of cortisol) and negative emotional interactions which shows just how sensitive schizophrenic symptoms are to social influences. It is possible that relapse can be largely curtailed, or at least

decreased, by interactions of low expressed emotion that do not participate in the feedback loop.

Yet, if anything I have also demonstrated the complexity of the issue. Families do not simply decide to interact in the styles they do, much of their interaction styles are strongly culturally mediated. More white Americans do interact with high expressed emotion than African Americans or Mexican Americans, but this is not because they do not care about their family members. Cultural influences, such as a focus on independence and self control, coupled with a biomedical orientation that creates an understanding of schizophrenia as permanent and defines the person as an uncontrolled “other” work in opposition to each other and probably contribute significantly to the greater amount of stressed interactions. In the United States, family members who are critical of their relatives’ behavior often see themselves as trying to help them learn how to control themselves and become more independent. They also perceive that they have lost their independence because of their role as caretakers, which increases their feelings of being burdened.

Interestingly, the studies on schizophrenia have shown that larger cultural influences can be seen to influence very particular human interactions and shape the behavioral and biological expression of the mental disorder in individuals. In Palau, the very expression of the disease seems strongly mediated by cultural factors with social instability and heightened stress (coupled with a malfunctioning stress response system) as the main culprits. The structure of the culture almost appears schizophrenogenic for men who are genetically predisposed to the disease. In Zanzibar, the sociocentric extended family focus, the concept of spirit possession, and the religious orientation toward duty and

devotion largely mediated interactions of low expressed emotion. Similarly, the focus on religious duty and the nurturing, sociocentric orientation of Mexican-Americans also influences interactions that create less stressful situations for schizophrenic individuals overall. These point to not only practices of family interaction, but also modes of understanding and coping with stress that help both family members and schizophrenic individuals manage the disease and improve their quality of life. However, in the case of Micronesian men and many schizophrenics in Western white households, the modes of understanding and cultural practices serve to exacerbate stress and therefore the illness severity. And perhaps these different cultural practices and philosophical frameworks also largely contribute to the likelihood that the illness develops at all. This is a particularly salient point in schizophrenic populations like the one in Micronesia, where the more stressed gender also has the higher prevalence of the mental illness.

From a perspective of medical anthropology, the question now is how can this knowledge be used to help schizophrenics? How can we increase interactions and larger cultural practices that are less conducive to the expression of schizophrenia? And how do we accomplish this without causing unforeseen harm? More importantly, is it even appropriate or possible to do this on a large scale? I don't think so, and until more studies have been done and there is more communication between the various disciplines trying to understand and help schizophrenic individuals, we simply do not know enough to create a plan of action with any degree of confidence. Hopefully, an increase in biocultural studies like the one conducted by Sullivan, Allen, and Nero (2007) will help to increase our understanding of not just the particular cultural effects on interaction but also the large scale cultural and biological influences.

FUTURE DIRECTIONS

It is important that more studies be done which connect the biological level of the structure and function of the schizophrenic brain with familial and community interactions and the larger cultural framework. I can think of a few directions to go in using Sullivan, Allen, and Nero's (2007) biocultural research model that also takes cortisol levels into account. One direction would be to simply study other populations with gendered disparities in the prevalence of the disorder that are greater than the worldwide average. In general, there is a significantly higher prevalence of schizophrenia among males as among females and the age of onset of symptoms (or first admission for psychotic symptoms) is five years earlier for males (Hart, Doherty, & Walsh, 2007; Murthey, et al. 1998). However, the average difference is small compared to the regional gender differences seen in areas like Palau, Micronesia.

The mean difference worldwide in age of onset for the sexes and sex differences in the prevalence of the disorder have been shown to change over time and regionally. Large variations from this mean are worth investigating because they could indicate the influence of social factors on biology. One area of divergence from the norm is in Bangalore, India where Murthey and colleagues (1998) found a very striking prevalence of schizophrenia among young women that ran contrary to the worldwide averages. They discussed their own and other studies conducted in India that found that there was no sex difference in age of onset of schizophrenia in India, while the age of onset for men was

not significantly different from the worldwide average. This means that women in India begin to present symptoms of the disease earlier than the average. More striking, even though the means were the same, the range was skewed toward the younger ages for the female subjects. This means that there was a larger number girls that first presented at 17 years or younger than boys, which is completely the opposite of the worldwide average. What is also interesting is that in the Murthey and colleagues (1998) subject group, women had significantly higher depression scores ($P < 0.04$). The researchers argued that, unlike other samples where men have an earlier onset rate than women, in India males are likely to be more sheltered from the stressors that would make them vulnerable to the disorder. They asserted that both sexes are similarly sheltered, rather than women being more sheltered than men.

I would go further and argue that men might indeed be more sheltered than women. The focus of Murthey and colleagues (1998) article was to present their findings and they very cursorily discussed possible cultural differences as contributing factors. However, if we treat the worldwide averages as based in biology, then deviations from that norm may be explained by environmental causes. For a future research study, it would be important to first establish variations in gender, depression and anxiety scores, age of onset, and overall prevalence of schizophrenia across regions in India. Murthey and colleagues (1998) treated their site as indicative of all of India, which is a mistake because India is comprised on many different regional, ethnic, and religious groups that have different cultural practices and beliefs. At the very least, examining regional differences in the expression of schizophrenia would be a necessary part of understanding the expression of the disorder in India. I would establish whether the differences found by Murthey and

colleagues (1998) were consistent across the country or were indicative of a few regions. Then I would establish a research pool in Bangalore or a region with similar outcomes and replicate the biobehavioral tests conducted by Sullivan, Allan, and Nero (2007). I would also include a cortisol saliva test to determine the basal cortisol levels and compare them between the gender and age groups. Preferably, I would have a healthy control group from the region from which I can gain baseline scores on all the tests I would run on the schizophrenic pool, which would include cortisol levels, smooth eye tracking scores, and behavioral test scores (which would include a Beck Depression Inventory to determine depression scores). I predict that there would be no bio-behavioral distinction in schizophrenic expression between my research group and the control population of schizophrenics that Sullivan, Allan, and Nero (2007) used from the United States. I also predict that the heightened depression scores for schizophrenic women found in the Murthey and colleagues (1998) study would be mirrored by heightened depression scores among the women in the local control group in comparison to their male counterparts. I would also be interested in establishing if there were sex differences in the severity of symptoms. This is a variable which I have not come across in the Indian schizophrenia literature.

I would also conduct an ethnographic analysis and a larger social and economic examination of the region in order to determine gender differences in the local life histories. From what I know about Indian marriage practices, the cultural practices surrounding men and women are largely the inverse of Palau, Micronesia. India is patrilineal and patrilocal and women leave their natal home to live with their husbands and the husband's family. They experience more stressed interactions between their natal

and marriage families than men do and receive less warmth. Women have little status until they prove their worth, whereas the status of men is largely stable and proscribed from birth. However, I would not be able to determine this as a causal factor until I did a more in-depth ethnographic analysis and compared it to my bio-behavioral results.

In their study of schizophrenia incidence in Ireland between 1971 and 2004, Hart, Doherty, and Walsh (2007) found that the incidence of first admission for schizophrenia (which is an indicator of overall incidence) has decreased dramatically between 1971 and 2004 with a much greater decline among women (702 to 291) than among men (926 to 514). Also the ages of first admission for schizophrenic symptoms were much higher than the worldwide average in Ireland (38 years rather than 25). However, they found a typical gendered relation to the onset of schizophrenia with men presenting 5 years earlier on average. More interesting to me, they discovered that marriage was protective. Individuals who had ever been married presented the onset of schizophrenia at a significantly higher age than those who had never been married. Marriage was also much more protective of men than women and was more predictive of age of onset than gender. Unfortunately, there were many unanswered questions concerning why the age of onset was so much higher in Ireland. It may be that 38 is not the actual age of onset, but that families are reluctant to admit newly psychotic relatives to the hospital, opting instead to care for them themselves until their symptoms become a major issue.

Nancy Scheper-Hughes (1979) in her ethnography of a rural Irish community and schizophrenia, found that there was a very different philosophy concerning the mentally ill in Ireland than in the United States or nearby England. She conducted a one-year study in County Kerry in 1976, which was a rural and economically depressed region of

Ireland. Schizophrenia in the County Kerry was at a very high rate compared to the rest of Ireland. During that time Ireland had three times as many schizophrenic patients in hospitals than England. Schizophrenia in Ireland was also much more prevalent in the western region and was strongly associated with being male, marginal agriculture, depopulation, isolation, celibacy, and unmarried status for men. In other words, it was associated with cultural and economic stressors.

Scheper-Hughes (1979) examined the ways in which locals viewed the schizophrenic individuals who lived in the community. She found that in some cases they were very well-tolerated and cared for by their families, but in other cases they were isolated and mistreated. She mentioned the distinction in Ireland, from ancient tradition, that the “fool” (mentally retarded usually) can participate in the community while the “lunatic” (the dangerous insane) is cast out of the community and either severely controlled or abandoned. This dichotomy still existed in the western Irish community. Some schizophrenics are seen as fools, but when they cross the line to lunatic, in other words when they prove to be dangerous in some capacity, they are shunned by the community. She found that schizophrenic individuals were often well tolerated by the whole community and often allowed to move freely through town. According to Scheper-Hughes, their families often cared for them in the home most if not all of their lives. However, the average hospital stay for schizophrenics was two years, which was and still is extremely long. Knowing the older age of first hospital admission for schizophrenics in Ireland, that has remained resilient and has even increased since the 1980s, it might be that most schizophrenics are not reported or hospitalized at first onset in Ireland. Instead, they may be handled by their families and the community until such a time that they have

an episode that cannot be handled. This could possibly explain the later date for first hospital admission for Ireland in general reported by Hart, Doherty, and Walsh (2007). However, the much longer hospital stays of the 1970s for schizophrenics could indicate that many of them found themselves in the 'lunatic' category and were cast off by their families.

Scheper-Hughes found a number of different cultural factors to explain why the incidence of schizophrenia was so high in Ireland and most of them had to do with stressful conditions which were related to cultural factors, such as sexual repression and a harshness and lack of intimacy between even close family members, and economic factors, such as joblessness and the exodus of women to find jobs and men with careers. Men, as Scheper-Hughes noted, were particularly stressed and had a much higher rate of schizophrenia than women in Ireland, then and now (Hart, Doherty, & Walsh, 2007; Scheper-Hughes, 1979).

For future research, I could replicate Sullivan, Allan, and Nero's (2007) study as mentioned above with a local control population to compare the biobehavioral data (psychological inventories, smooth eye tracking, etc.). I would also obtain basal cortisol samples between men and women, according to marital status, and between the schizophrenic and control groups. I would however, need to develop more reliable criteria for onset of schizophrenia if indeed individuals are not admitted to the hospital and diagnosed until long after first onset. Yet, if first onset is truly that late then it would also be necessary to determine why this is. However, since the overall incidence of schizophrenia in Ireland is still high, I doubt that first onset will be significantly later than that of other regions. I would predict that, as Scheper-Hughes found, unmarried men

would have the higher basal cortisol levels, higher incidence of the disease, and a greater severity because of differences in interactions styles of family members and cultural and economic pressures. I also predict that unmarried men would score higher on the Beck Depression Inventory, if the same conditions which Scheper-Hughes described still exist. I would then conduct an ethnography to determine how much the culture of rural Ireland had changed since Scheper-Hughes' study and to gather information concerning interaction styles and cultural practices and their underlying beliefs that related to schizophrenic individuals.

Another interesting area for further study is among migrant groups. In a metastudy of schizophrenia prevalence rates by gender, 'development' status, migrant status, or other special statuses conducted by Saha and colleagues (2005), the researchers found that there was no worldwide gender difference in the prevalence of the disorder. However, in a similar metastudy of schizophrenia prevalence, McGrath and colleagues (2004) found that there was a significantly higher incidence of schizophrenia among men than women. However, sex differences can vary greatly in different pockets and these disparities are worth studying to determine if they are indicative of a worldwide average or other local features such as economic and social factors. The study conducted by Saha and colleagues (2005) also supported the higher prevalence of schizophrenia in 'developed' countries as opposed to 'developing' countries. Interestingly, both studies (Saha et al., 2005; McGrath et al., 2004) also supported a higher prevalence rate of schizophrenia within immigrant populations when compared to native populations.

This finding points to a fascinating area for future research. Why is there a higher prevalence of schizophrenia among immigrant populations? Is it because of cultural

differences and prejudices influencing psychiatrists' diagnoses or is there something about being an immigrant that makes a person more susceptible? I would argue that cultural biases might come into play when diagnoses are given, but that being an immigrant can indeed increase an individual's vulnerability in the prodromal period. The role of stress and a malfunctioning hippocampus and HPA axis in the prodromal period has been established and it seems likely to me that migrant individuals experience more stress on average than a native does. It would be interesting to conduct a study comparing an immigrant population and a native population within the same city. The overall prevalence could be studied and this could be controlled for by socioeconomic status. If there is a significantly greater prevalence of schizophrenia among immigrants, I would then conduct a study to determine if stress is a causative factor. There would be two sets of independent variables that are most important to this study: the immigrant/native status and the schizophrenic/healthy control status. First, the basal cortisol levels would be taken of both the schizophrenic and control groups to establish a baseline difference. I would predict from previous studies, that the basal cortisol levels among the schizophrenic group would be higher. Then, I would separate the results of the immigrant/schizophrenic, native/schizophrenic, immigrant/control, and native/control groups. If stress is a major factor for the differential prevalence of schizophrenia among immigrants, then their basal cortisol levels should be higher in the control group, if not both groups. I would also want to do a cultural study that examines the family life of schizophrenics in both the native and migrant groups (controlling for economic status) in order to determine differences in interaction styles. I would expect that a more stressed control group would lead to more stressed (i.e. high expressed emotion) interactions at

home between healthy family members and their schizophrenic relatives. I would also examine if there are differences in the way mental illness is perceived and cultural conventions and beliefs that guide interactions. It would be easier to study one immigrant population from one culture and compare it to a native group, but examining many different immigrant groups could also be very enlightening concerning the impact of stress and similarities across different cultural groups.

There are many avenues to explore the impact cultural factors have on the onset of schizophrenia and the behavioral symptomology and biological processes of this mental disorder. Most striking are areas of incongruence between populations such as striking gender differences, differences in marital status, and immigrant status. Very few biocultural studies have been done on schizophrenia and so it is a very open and fertile field of inquiry with many questions yet unanswered.

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